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L3 ANSWER 11 OF 23 MEDLINE
AN 2000168638 MEDLINE
DN 20168638 PubMed ID: 10706121
TI Enhancement of DNA vaccine potency by linkage of antigen gene to an HSP70 gene.
AU Chen C H; Wang T L; Hung C F; Yang Y; Young R A; Pardoll D M; Wu T C
CS Department of Oncology, The Johns Hopkins Medical Institutions, Baltimore, Maryland 21287, USA.
NC 5 PO1 34582-01 (NCI)
RO1 CA72631-01 (NCI)
U19 CA72108-02
SO CANCER RESEARCH, (2000 Feb 15) 60 (4) 1035-42.
Journal code: 2984705R. ISSN: 0008-5472.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; AIDS
EM 200003
ED Entered STN: 20000330
Last Updated on STN: 20000330
Entered Medline: 20000320
AB Nucleic acid vaccines represent an attractive approach to generating antigen-specific immunity because of their stability and simplicity of delivery. However, there is still a need to increase the potency of DNA vaccines. Using human **papillomavirus** type 16 E7 as a model antigen, we evaluated the effect of linkage to Mycobacterium tuberculosis **heat shock protein** 70 (HSP70) on the potency of antigen-specific immunity generated by naked DNA vaccines. We found that vaccines containing E7-HSP70 fusion genes increased the frequency of E7-specific CD8+ T cells by at least 30-fold relative to vaccines containing the wild-type E7 gene. More importantly, this fusion converted a less effective vaccine into one with significant potency against established E7-expressing tumors. Surprisingly, E7-HSP70 fusion vaccines exclusively targeted CD8+ T cells; immunological and antitumor effects were completely CD4-independent. These results indicate that fusion of HSP70 to an antigen gene may greatly enhance the potency of DNA vaccines via CD8-dependent pathways.
CT Check Tags: Animal; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
*Bacterial Proteins: GE, genetics
CD8-Positive T-Lymphocytes: IM, immunology
*Cancer Vaccines: IM, immunology
*Heat-Shock Proteins 70: GE, genetics
Killer Cells, Natural: IM, immunology
Mice
Mice, Inbred C57BL
*Oncogene Proteins, Viral: GE, genetics
Oncogene Proteins, Viral: IM, immunology
Vaccination
*Vaccines, DNA: IM, immunology
CN 0 (Bacterial Proteins); 0 (Cancer Vaccines); 0 (Heat-Shock Proteins 70); 0 (Oncogene Proteins, Viral); 0 (Vaccines, DNA); 0 (oncogene protein E7-human **papillomavirus** type 16)

L3 ANSWER 12 OF 23 MEDLINE
AN 2000148983 MEDLINE
DN 20148983 PubMed ID: 10684306
TI Recombinant adeno-associated virus expressing human **papillomavirus** type 16 E7 peptide DNA fused with **heat shock protein** DNA as a potential vaccine for cervical cancer.

AU Liu D W; Tsao Y P; Kung J T; Ding Y A; Sytwu H K; Xiao X; Chen S L
CS Department of Microbiology and Immunology, Taipei, Taiwan, Republic of China.
SO JOURNAL OF VIROLOGY, (2000 Mar) 74 (6) 2888-94.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; AIDS
EM 200004
ED Entered STN: 20000413
Last Updated on STN: 20000413
Entered Medline: 20000403
AB In this study, we explore a potential vaccine for human **papillomavirus** (HPV)-induced tumors, using **heat shock protein** as an adjuvant, a peptide vaccine for safety, and adeno-associated virus (AAV) as a gene delivery vector. The tumor vaccine was devised by constructing a chimeric gene which contained HPV type 16 E7 cytotoxic T-lymphocyte (CTL) epitope DNA (M. C. Feltkamp, H. L. Smits, M. P. Vierboom, R. P. Minnaar, B. M. de Jongh, J. W. Drijfhout, J. ter Schegget, C. J. Melief, and W. M. Kast, Eur. J. Immunol. 23:2242-2249, 1993) fused with the **heat shock protein** gene as a tumor vaccine delivered via AAV. Our results demonstrate that this vaccine can eliminate tumor cells in syngeneic animals and induce CD4- and CD8-dependent CTL activity in vitro. Moreover, studies with knockout mice with distinct T-cell deficiencies confirm that CTL-induced tumor protection is CD4 and CD8 dependent. Taken together, the evidence indicates that this chimeric gene delivered by AAV has potential as a cervical cancer vaccine.
CT Check Tags: Animal; Female; Human; Support, Non-U.S. Gov't
Blotting, Northern
CD4-Positive T-Lymphocytes: IM, immunology
CD8-Positive T-Lymphocytes: IM, immunology
*Cancer Vaccines: IM, immunology
Cell Line, Transformed
*Cervix Neoplasms: PC, prevention & control
DNA, Viral
*Dependovirus
Dependovirus: GE, genetics
Epitopes, T-Lymphocyte: GE, genetics
Epitopes, T-Lymphocyte: IM, immunology
Gene Fusion
Genetic Vectors: GE, genetics
Heat-Shock Proteins 70: GE, genetics
*Heat-Shock Proteins 70: IM, immunology
Mice
Mice, Inbred C57BL
Mice, Knockout
Muscle, Skeletal
Oncogene Proteins, Viral: GE, genetics
*Oncogene Proteins, Viral: IM, immunology
Papillomavirus, Human: GE, genetics
***Papillomavirus, Human: IM, immunology**
Peptides: GE, genetics
Peptides: IM, immunology
T-Lymphocytes, Cytotoxic: IM, immunology
*Vaccines, DNA: IM, immunology
Vaccines, Synthetic: GE, genetics
Vaccines, Synthetic: IM, immunology
*Viral Vaccines: IM, immunology
CN 0 (Cancer Vaccines); 0 (DNA, Viral); 0 (Epitopes, T-Lymphocyte); 0 (Genetic Vectors); 0 (Heat-Shock Proteins 70); 0 (Oncogene Proteins,

L10 ANSWER 8 OF 9 MEDLINE
AN 93100820 MEDLINE
DN 93100820 PubMed ID: 7677955
TI Progression from papilloma to carcinoma is accompanied by changes in antibody response to **papillomavirus** proteins.
AU Lin Y L; Borenstein L A; Selvakumar R; Ahmed R; Wettstein F O
CS Department of Microbiology and Immunology, School of Medicine, University of California, Los Angeles 90024.
NC CA 50339 (NCI)
SO JOURNAL OF VIROLOGY, (1993 Jan) 67 (1) 382-9.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199301
ED Entered STN: 19930205
Last Updated on STN: 19960129
Entered Medline: 19930115
AB Cottontail rabbit **papillomavirus** induces benign tumors, papillomas, in rabbits which progress at a high frequency to malignant tumors, carcinomas. Cottontail rabbit **papillomavirus** therefore provides an experimental model for oncogenic human papillomaviruses. The nature of the antigens recognized by the host has not been identified at any stage of tumor development. Here, we characterized the humoral **immune response** to viral antigens in cottontail and domestic rabbits at the papilloma stage, in domestic rabbits at the carcinoma stage, and in animals in which papillomas had regressed. Antibodies to linear epitopes were identified by Western blotting (immunoblotting) with bacterial fusion proteins, and evidence for recognition of conformational epitopes was obtained by immunoprecipitation. An **immune response** to the **early proteins** E1, E2, E6, and E7 was detected only in a fraction of the animals, and all animals were negative for E4 and E5. The response to E6 and E7 peaked around 7 months and then decreased, while that to E1 and E2 remained level after an initial raise. The antibody response to structural proteins was low at the papilloma stage, and antibodies to L1 recognized predominantly conformational epitopes. As papillomas progressed to carcinomas, there was a drastic increase in the response to L1 and L2, suggesting a change in interaction between virus-infected host cells and the host's immune system.
CT Check Tags: Animal; Comparative Study; Support, U.S. Gov't, P.H.S.
Antibodies, Viral: IM, immunology
Antibody Formation
Carcinoma: ET, etiology
*Carcinoma: IM, immunology
*Cell Transformation, Neoplastic: IM, immunology
Epitopes: IM, immunology
*Papilloma: IM, immunology

Viral); 0 (Peptides); 0 (Vaccines, DNA); 0 (Vaccines, Synthetic); 0 (Viral Vaccines); 0 (oncogene protein E7-human **papillomavirus** type 16)

L3 ANSWER 13 OF 23 MEDLINE
AN 1999333754 MEDLINE
DN 99333754 PubMed ID: 10385551
TI An immunohistochemical analysis of **heat shock protein** 70, p53, and estrogen receptor status in carcinoma of the uterine cervix.
AU Park C S; Joo I S; Song S Y; Kim D S; Bae D S; Lee J H
CS Samsung Medical Center, School of Medicine, Sung Kyun Kwan University, 50 Ilwon-dong, Kangnam-ku, Seoul, 135-710, Korea.
SO GYNECOLOGIC ONCOLOGY, (1999 Jul) 74 (1) 53-60.
Journal code: 0365304. ISSN: 0090-8258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199908
ED Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990816
AB OBJECTIVES: It has been shown that heat shock proteins (HSPs) protect cells from death caused by various noxious stimuli. Overexpression of HSP70 seems to be related to hormonal regulation of cell proliferation and/or down-regulation of sex steroid receptors. Wild-type p53 has been reported to repress HSP70 gene expression. It has been shown that mutant p53-HSP70 complex is highly expressed in cancer. However, the relationship between HSPs and steroid receptors or tumor suppressor gene products has not been well understood in uterine cervical carcinoma. This study was undertaken to examine the expression of HSP70, estrogen receptor (ER), and p53 in carcinoma of the uterine cervix. In addition, we analyzed HPV infection status and compared it to such immunohistochemical parameters. We also analyzed the relationship between these biological products and their clinicopathologic characteristics. METHODS: Paraffin-embedded tissue sections were obtained from 84 patients with carcinoma of the uterine cervix. Expression of HSP70, p53, and ER was evaluated by immunohistochemical staining using anti-HSP70 monoclonal antibody (SPA810), anti-p53 (BP53.12), and ER1D5 antibody, respectively. PCR HPV detection was done by dot hybridization method. RESULTS: Positive staining of HSP70 was detected in 73% of the cases. HSP70 positivity was significantly higher in stage I cervical cancer than in stages II-IV ($P = 0.02$). This was associated with neither tumor size, lymph node status, parametrial involvement status, nor tumor markers (TA-4). Furthermore, there was no significant correlation between HSP70 positivity and the expression of p53 or ER or HPV infection status. CONCLUSION: These data suggested that HSP70 positivity was frequent in uterine cervical cancer, especially in the early stages. However, this was not significantly correlated with clinicopathologic characteristics nor with the expression of p53 or ER nor with HPV infection in carcinoma of the uterine cervix.

CT Check Tags: Female; Human
*Cervix Neoplasms: CH, chemistry
Cervix Neoplasms: PA, pathology
Cervix Neoplasms: VI, virology
*Heat-Shock Proteins 70: AN, analysis
Immunohistochemistry
Papillomavirus, Human: IP, isolation & purification
Papovaviridae Infections: VI, virology
*Protein p53: AN, analysis
*Receptors, Estrogen: AN, analysis
Tumor Virus Infections: VI, virology

CN 0 (Heat-Shock Proteins 70); 0 (Protein p53); 0 (Receptors, Estrogen)

L3 ANSWER 17 OF 23 MEDLINE
AN 96013135 MEDLINE
DN 96013135 PubMed ID: 7556594
TI HPV16 E7 oncoprotein induces expression of a 110 kDa **heat shock protein**.
AU Morozov A; Subjeck J; Raychaudhuri P
CS Department of Biochemistry (M/C 536), University of Illinois at Chicago 60612, USA.
SO FEBS LETTERS, (1995 Sep 11) 371 (3) 214-8.
Journal code: 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-L40406
EM 199510
ED Entered STN: 19951227
Last Updated on STN: 19951227
Entered Medline: 19951026
AB **Heat shock protein** genes are induced by various kinds of stress. Besides stress, the heat shock family gene hsp70 has been shown to be induced by growth-stimulating agents such as the DNA virus oncoproteins and serum. Here, we report cloning of a novel cDNA that encodes a 100 kDa **heat shock protein**-related polypeptide as a human **papillomavirus** oncoprotein E7-inducible gene. E7 induces expression of this **heat shock protein** at the level of RNA synthesis. Moreover, the induction of this **heat shock protein**-mRNA was dependent on the conserved region 2 of the E7 protein, which is essential for binding to the proteins of the retinoblastoma family.
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't
3T3 Cells
Amino Acid Sequence
Base Sequence
Cloning, Molecular
DNA, Complementary
Gene Expression Regulation
Heat-Shock Proteins 70: BI, biosynthesis
*Heat-Shock Proteins 70: GE, genetics
Heat-Shock Response
Mice
Molecular Sequence Data
Mutation
*Oncogene Proteins, Viral: PH, physiology
RNA, Messenger: ME, metabolism
Recombinant Proteins
Sequence Homology, Amino Acid
CN 0 (DNA, Complementary); 0 (Heat-Shock Proteins 70); 0 (Oncogene Proteins, Viral); 0 (RNA, Messenger); 0 (Recombinant Proteins); 0 (**heat-shock protein** 110); 0 (oncogene protein E7-human **papillomavirus** type 16)